

# Fetal Monitoring



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One of the most important goals for the anesthesiologist caring for a pregnant woman should be to maintain the uteroplacental unit and fetus in optimal condition. Moreover, knowledge of the fetal condition and activity of the uterus can have important implications for the anesthetic plan. Hence, an adequate knowledge of uterine and fetal monitoring is important.

## Antepartum Monitoring

Assessment of the fetal condition prior to the onset of labor may affect the timing and mode of delivery.

## Biophysical Profile

Antepartum evaluation of the high-risk fetus is possible by evaluation of five biophysical activities, most of which are assessed by ultrasound:

1. Fetal movement (three gross body or limb movements in 30 min)
2. Fetal tone (at least one extension of an extremity and return to flexion)
3. Fetal breathing movements (at least one episode 30 s in 30 min)
4. Heart rate activity (a nonstress test, see below)
5. Volume of amniotic fluid (at least 2 cm pocket of fluid observable)

The first four parameters reflect the presence of normal fetal central nervous system activity, whereas amniotic fluid volume is an indicator of long-term or chronic fetal condition. These parameters are all measured by ultrasound except for the fetal heart rate. The variables are scored 2 if normal and 0 if abnormal. A score of 8 or 10 is normal, and strongly predicts a healthy newborn with a false-negative rate of less than 0.1%.<sup>1</sup> A score of 0, conversely, is strongly correlated with fetal death or neonatal morbidity, and is considered an obstetric emergency.<sup>2</sup> Intermediate scores have equivocal predictive ability, although a score of 4 or below is considered abnormal and is often treated as indication for delivery.

## Nonstress Test (NST)

This involves the detection of changes in the fetal heart rate and fetal movement in association with uterine contraction. The fetal heart rate is monitored by Doppler for 20 min (the test can be extended for additional 20 min periods if the fetus is in a sleep cycle). The test is described as reactive (normal) if there are two fetal movements in 20 min with accelerations of the fetal heart rate of at least 15 BPM for at least 15 s. The test is described as nonreactive in the absence of fetal movement or accelerations of the fetal heart rate. Early gestational age, fetal sleep, and maternal smoking can cause a nonreactive test in otherwise normal fetuses. However, a reactive NST is reassuring: stillbirth within one week of a normal test was seen

in only 0.2% of cases in a large observational study.<sup>3</sup> On the other hand, the stillbirth rate after a nonreactive test was just 2.6%, indicating a very high false-positive rate.

### Contraction Stress (Oxytocin Challenge) Test

In the presence of a nonreactive NST or other concern for fetal well-being, the contraction stress test (CST), sometimes called the oxytocin challenge test (OCT) may be employed. Intravenous oxytocin is infused to induce three adequate uterine contractions within a 10-min period. *The oxytocin challenge test is contraindicated if there is history of classical cesarean section, placenta previa, high risk of premature labor, or preterm premature rupture of membranes.* The oxytocin challenge test is considered to be positive (nonreassuring) if persistent late decelerations occur in at least 50% of contractions. The test is interpreted as negative (reassuring) in the presence of normal fetal heart rate tracings without late or significant variable decelerations. Other patterns are graded as equivocal or unsatisfactory. Positive tests predict abnormal heart rate patterns in labor and may be an indication for cesarean delivery.

### Doppler Velocimetry

Ultrasound examination of blood flow through the umbilical artery towards the placenta can detect conditions of high placental vascular resistance, which correlates with uteroplacental insufficiency and fetal compromise. An elevation in the systolic to diastolic velocity ratio, or detection of absent or reversed diastolic flow, is associated with a higher incidence of perinatal death.<sup>4</sup> Especially in cases of absent or reversed diastolic flow, prompt delivery, usually by cesarean, is indicated unless the fetus is very premature.

### Assessment of Fetal Lung Maturity

Tests of amniotic fluid components can be used to assess maturity of the fetal lung sufficient to avoid the neonatal respiratory distress syndrome. Phospholipids, the major components

of lung surfactant, are produced by fetal alveolar cells in a sufficient amount by 36 weeks' gestation but may not be present in sufficient amounts at earlier gestational ages.

Several tests are available. The lecithin/sphingomyelin ratio (L/S) was developed in the 1970's to predict fetal lung maturity and is normal when the ratio is 2 in uncomplicated pregnancies. For diabetic parturients, the ratio should be at least 3.5 or higher. Measurements of saturated phosphatidylglycerol are occasionally used in normal parturients; the normal value is 500 mg/dL, whereas in diabetics it is 1,000 mg/dL. Both of these classic tests are cumbersome, require a clean, uncontaminated sample of amniotic fluid, and are subject to substantial inter-laboratory variation.

A simpler and faster test relies on the fluorescence generated by polarized light when a specific probe for surfactant is mixed with amniotic fluid. This test, the TD<sub>X</sub>-FLM (fetal lung maturity), has become much more popular. It is expressed in milligrams of surfactant per gram of albumin. A FLM value of <40 mg/g indicates immature lung, and above 55 mg/g (60 in some institutions) predicts adequate lung maturity. Intermediate values, unfortunately, are considered indeterminate and have poor predictive ability. Advantages of the FLM are the ability to use a vaginal pool sample in patients with ruptured membranes, its simplicity and rapidity, and its consistency across laboratories. In addition, it has proven reliable at the usual cutoff values in diabetic patients.<sup>5</sup>

## **Uterine Contraction Monitoring**

Assessment of uterine activity is important in predicting the normal progress of labor and also fetal well-being. Parameters describing uterine activity are (1) baseline uterine tone and amplitude, (2) duration of contractions, and (3) the interval between contractions. Normal baseline tone varies between 8 mmHg and 20 mmHg and increases to between 25 mmHg and 75 mmHg during contractions. However, the peak pressure can rise to 130 mmHg with bearing-down efforts in the second stage of labor. A contraction can last from 30 s to 90 s, and the interval between contractions normally varies from 2 min to 3 min.

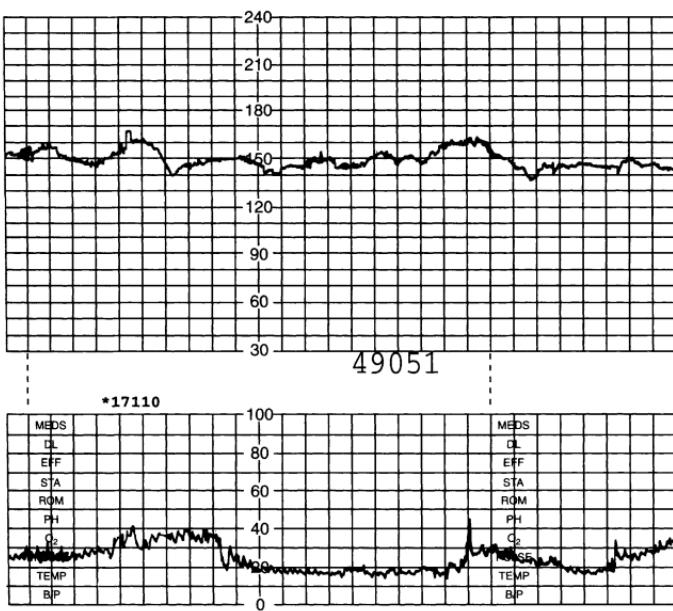
A tocodynamometer qualitatively measures uterine activity when placed on the skin over the uterus by detecting the ease of indenting the abdominal wall, which decreases during the muscular contraction of the uterus.<sup>6</sup> However, one of the major limitations of external tocodynamometry is inaccuracy from variability in positioning of the instrument, tension of the bands securing it, and distensibility of the abdominal wall. Internal monitoring is more accurate and reliable, as it uses either a fluid-filled catheter in the uterus connected to a pressure transducer, or a direct pressure sensor placed on the end of a probe placed in the uterus. Internal monitoring requires (1) engagement of the presenting part, (2) adequate cervical dilatation, and (3) ruptured membranes. Internal measurement of uterine activity is more commonly used in high-risk cases (e.g., diabetes, postmaturity), when monitoring externally is technically difficult (e.g., morbid obesity), or when quantitative documentation of adequacy of uterine contraction is necessary. Comparative studies, however, have not demonstrated any clinically relevant advantage of internal monitoring.<sup>7</sup>

## Fetal Heart Rate Monitoring

The fetal heart rate (FHR) can be monitored by an external probe that uses Doppler ultrasound to detect fetal heartbeats, or by direct application of a bipolar electrode to the fetal presenting part. In either case, the FHR is plotted against time on a strip recorder and inspected for baseline heart rate, variability, and periodic changes, including decelerations and accelerations. The definitions of these terms have recently been reviewed and clarified in an effort to standardize terminology and improve predictive ability of the technique.<sup>8</sup> FHR monitoring is very widely used in US labor units and many other countries worldwide. Remarkably, numerous randomized controlled trials have failed to show significant improvements in fetal condition at birth or a reduction in cerebral palsy or perinatal death.<sup>9</sup>

### Baseline Heart Rate

*The normal baseline fetal heart rate varies between 110 and 160 beats per minute (BPM), and it is modulated by parasympathetic and sympathetic nerve activity (Fig. 11-1). The*



**Figure 11-1.** Normal fetal heart rate pattern.

baseline is determined by inspection and should not include periodic changes (accelerations and decelerations).

*Fetal tachycardia* is diagnosed when the baseline exceeds 160 BPM.<sup>8</sup> The major causes include:

1. Fetal hypoxia
2. Maternal fever, most often associated with infection
3. Maternal administration of sympathomimetic drugs, e.g., ephedrine,  $\beta$ -mimetic drugs for tocolysis (terbutaline), epinephrine
4. Maternal administration of parasympatholytic drugs, e.g., atropine, phenothiazines
5. Maternal hyperthyroidism
6. Fetal anemia
7. Fetal tachydysrhythmias

*Fetal bradycardia* is defined as a fetal heart rate less than 110 BPM.<sup>8</sup> The major causes include:

1. Prolonged fetal hypoxia
2. Fetal head or umbilical cord compression

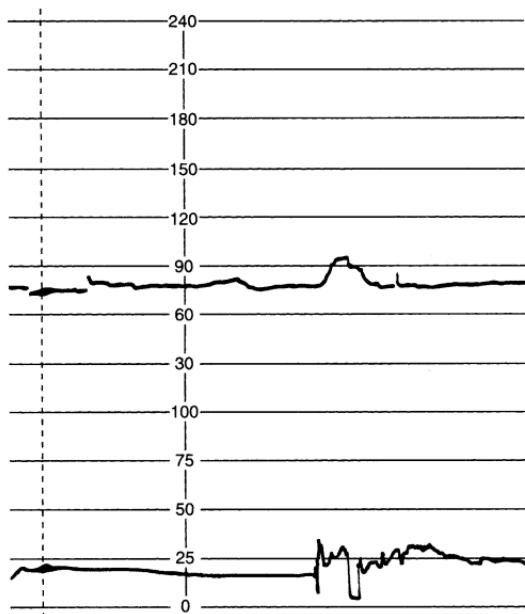
3. Maternal administration of parasympathomimetic drugs, e.g., neostigmine
4. Maternal administration of  $\beta$ -adrenergic antagonists, e.g., propranolol
5. Fetal congenital heart block
6. Combined spinal epidural technique
7. Prolonged maternal hypotension

## Baseline Variability

*Baseline variability is generally recognized as the single most important parameter for the recognition of intrauterine fetal well-being.* Baseline variability is due to a constant battle between the fetal sympathetic (increasing the heart rate) and parasympathetic (decreasing the heart rate) systems. The presence of good baseline variability is an indicator of intact central nervous system as well as normal cardiac functions.

Variability was previously classified into short-term variability, representing beat-to-beat differences of 5–15 beats, and long-term variability, fluctuations with a frequency of 3–5 cycles per minute. *This nomenclature is no longer considered valid.* Variability is now classified as absent, minimal (less than 5 BPM), moderate (6–25 BPM), and marked ( $>25$  BPM).<sup>8</sup> Minimal or absent variability (Fig. 11-2) is a strong predictor of neonatal acidosis<sup>10</sup> but there are other causes of diminished variability so the false-positive rate is high. Various factors that can affect variability include:

1. Fetal hypoxemia
2. Maternally administered opioids
3. Maternally administered sedatives and hypnotics, e.g., barbiturates, benzodiazepines, phenothiazines
4. Maternally administered parasympatholytic drugs, e.g., atropine or phenothiazines
5. Fetal sleep
6. Inhalation general anesthesia<sup>11</sup>
7. Extreme prematurity
8. Fetal tachycardia, fetal heart block
9. Maternally administered magnesium sulfate
10. Maternal sepsis



**Figure 11-2.** Decreased variability of the fetal heart rate.

### Fetal Heart Rate Pattern (Periodic Changes)

Periodic changes are defined as transient accelerations or decelerations of the fetal heart rate of short duration followed by a return to baseline levels. Recurrent decelerations accompany more than half of uterine contractions; intermittent changes occur in less than half.<sup>8</sup> There are three categories of decelerations: early, variable, and late.

#### *Early Decelerations*

Characteristics of early decelerations (Fig. 11-3) are as follows:

1. Symmetrical, U-shaped deceleration
2. Gradual onset and slow return to baseline (>30 s onset to nadir of FHR)

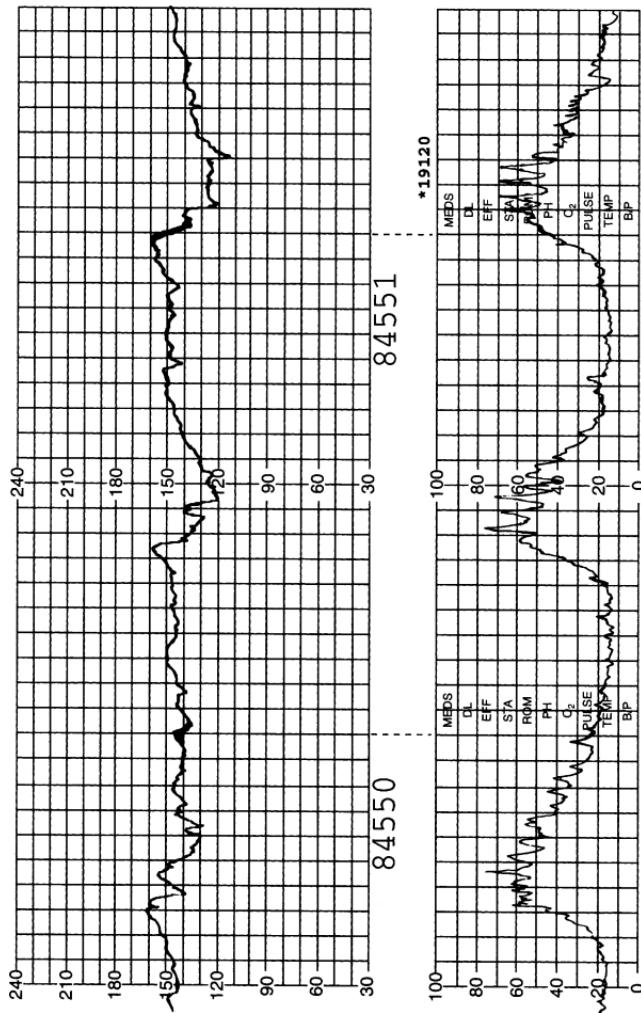
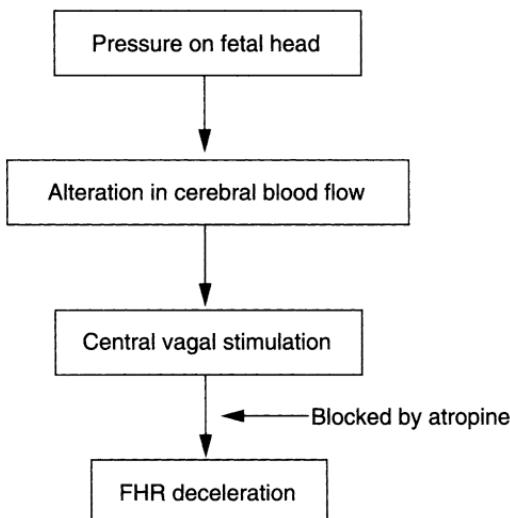


Figure 11-3. Early decelerations.

3. Mirror image of uterine contraction in duration and timing of peak change in HR with peak of contraction corresponding to nadir of FHR
4. Decrease in HR generally not more than 20–30 BPM



**Figure 11-4.** Mechanism of early decelerations (FHR = fetal heart rate). (Adapted from Freeman.<sup>17</sup>)

Two mechanisms for early deceleration that have been suggested are (1) fetal head compression with increased intracranial pressure (Fig. 11-4) and (2) increased volume of blood entering the fetal circulation during contractions, thus triggering baroreceptor reflex activity. Both of these mechanisms are vagally mediated and can be prevented by atropine.<sup>12</sup>

### *Variable Decelerations*

*This is the most common of all fetal heart rate patterns* (Fig. 11-5), and the characteristics of this pattern are as follows:<sup>8</sup>

1. Abrupt onset and return to baseline (<30 s)
2. Decrease  $\geq 15$  BPM, duration  $\geq 15$  s, duration <2 min
3. Variability in duration, shape, size, and timing relative to successive contractions
4. May be accompanied by brief accelerations ("shoulders") before and after departure from baseline

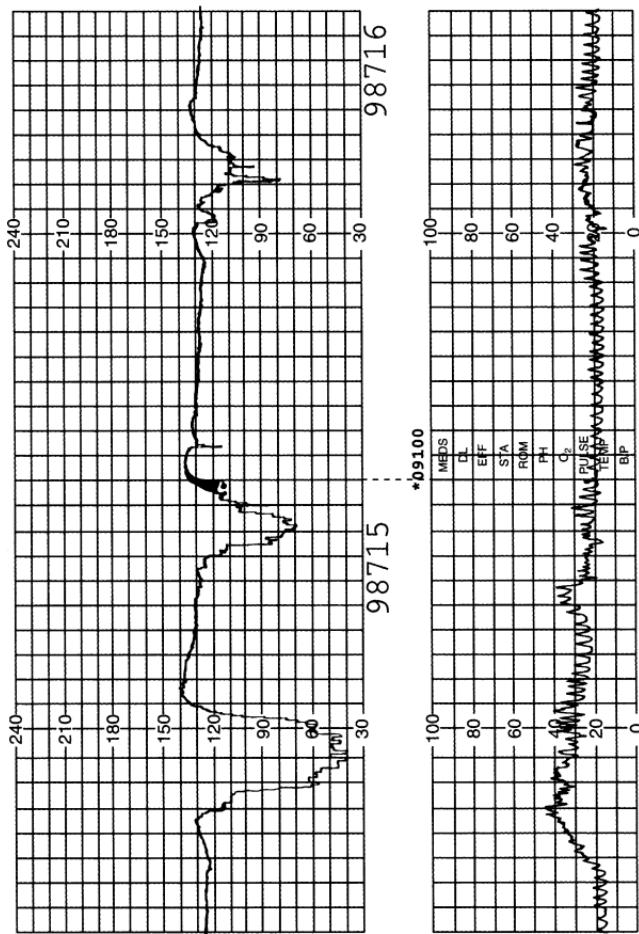


Figure 11-5. Variable decelerations.

Variable decelerations are usually caused by compression of the umbilical cord against fetal body parts, (e.g., head, neck, or shoulder). Because the maximum pressure on the cord is generated at the time of uterine contractions, the deceleration coincides with uterine contraction, but due to changes in relative fetal and cord position they vary in morphology.

They are likely mediated by vagal output. Depending upon the magnitude of the decrease in the fetal heart rate, variable decelerations have been further subdivided into (1) mild (duration less than 30 s and deceleration not below 80 BPM), (2) moderate (regardless of the duration, the fetal heart rate is less than 80 BPM), and (3) severe (duration greater than 60 s and a fetal heart rate less than 70 BPM). However, these classifications have not been tested for predictive validity.<sup>8</sup>

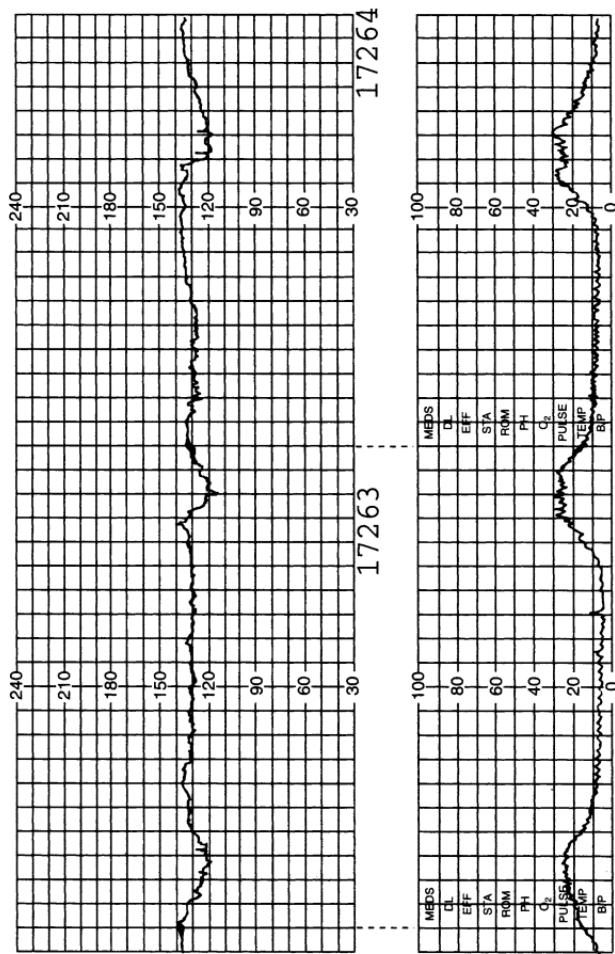
### *Late Decelerations*

Late decelerations (Fig. 11-6) are characterized by the following:

1. Symmetrical gradual onset and return to baseline ( $\geq 30$  s from onset to nadir).
2. Onset and nadir lag beginning and peak of uterine contraction (typically  $\geq 30$  s)
3. Return to baseline after the end of the associated uterine contraction
4. Decrease in FHR is usually mild (10–20 BPM) and rarely more than 30–40 BPM.

There is some correlation between the frequency of late decelerations and the degree of fetal hypoxia. Moderate variability in the setting of late decelerations is somewhat less concerning than diminished or absent variability with the same decelerations.<sup>8</sup> The major cause of late decelerations is reduced placental perfusion, as can be seen during hypotension (e.g., following regional analgesia), aortocaval compression, placental abruption, maternal diabetes mellitus, preeclampsia, and post-dates pregnancy.

Besides these three main patterns, the other patterns that have been described are prolonged decelerations and a sinusoidal pattern. *Prolonged decelerations* are decreases in HR  $\geq 15$  BPM which last  $\geq 2$  min but  $< 10$  min. Longer decelerations are considered baseline changes. Prolonged decelerations are considered concerning but not as ominous as recurrent late or variable decelerations with absent variability. A *sinusoidal pattern* is associated with a sine-wave pattern above and below the baseline with a frequency of 3–5 per min. A benign



**Figure 11–6.** Late decelerations. (Adapted from Martin.<sup>18</sup>)

sinusoidal pattern has been associated with opioid agonist-antagonist drugs (e.g., butorphanol). A major obstetric cause is severe fetal anemia usually associated with Rh incompatibility. In this setting, the sinusoidal pattern is considered ominous.<sup>8</sup>

## Other Modalities

### Fetal Scalp Blood Sampling

Fetal scalp blood pH determination is a direct way to test for the presence of acidemia. A sample of capillary blood is obtained by a small incision in the fetal scalp and analyzed for pH, typically in duplicate or triplicate. Normal fetal scalp pH varies between 7.25 and 7.32; mild acidemia is documented when the pH varies between 7.20 and 7.24; and severe acidemia is noted when the pH is less than 7.20. Unfortunately, the sensitivity and specificity of the test in predicting umbilical blood pH at delivery is modest.<sup>13</sup> Furthermore, scalp blood pH laboratories are expensive and volume is not generally high enough to justify the cost for the vast majority of labor units. Thus the popularity of the technique has sharply declined over the last decade.<sup>14</sup>

### Fetal Pulse Oximetry

In theory, continuous measurement of fetal oxygenation should provide minute-by-minute information of fetal well being, because ultimately poor oxygenation is the cause of most fetal compromise during labor. A modified pulse oximeter uses a reflectance probe, generally placed alongside the fetal cheek or temple. Unfortunately, the technique has not proven to reduce interventions for presumed fetal compromise, including cesarean delivery, nor has its use improved fetal condition at birth.<sup>15</sup> ACOG does not endorse its use at this time.<sup>16</sup>

## Implications for Anesthesia Care

The anesthesiologist should be aware of fetal assessment for several reasons. First, the timing and urgency of delivery are often dictated by the results of fetal monitoring. Awareness of a deteriorating FHR pattern or poor biophysical profile can alert the anesthesiologist to prepare for urgent delivery, or to encourage a parturient and the obstetric caregivers to initiate regional anesthesia in anticipation of emergency delivery.

Second, decelerations and other nonreassuring FHR tracings may require obstetrical interventions for ultimate resolution but the anesthesiologist may be called on to seek and remedy reversible causes. This may include proper positioning to avoid aortocaval compression, use of supplemental oxygen, correction of hypotension, intravenous fluid bolus, and rarely maneuvers to reduce uterine tone (e.g., intravenous nitroglycerin). Finally, enhanced communication between the obstetrical, nursing, and anesthesiology providers is facilitated by a common understanding of the factors influencing the formation of the obstetrical care plan.

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